A COMPARATIVE STUDY ON THE RISK FACTOR, CLINICAL PROFILE, AND OUTCOME OF ACUTE MYOCARDIAL INFARCTION IN PATIENTS OF AGE LESS THAN AND MORE THAN 40 YEARS

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BONAFIDE CERTIFICATE

Certified that the dissertation titled **"A COMPARATIVE STUDY ON THE RISK FACTOR, CLINICAL PROFILE, AND OUTCOME OF ACUTE MYOCARDIAL INFARCTION IN PATIENTS OF AGE LESS THAN AND MORE THAN 40 YEARS"** is a bonafide work of the candidate **Dr.M.GEETHA,** post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai – 10, done under my guidance and supervision, in partial fulfillment of regulations of **The Tamilnadu Dr. MGR Medical University** for the award of M.D. Degree Branch I, (General Medicine) during the academic period from May 2007 to March 2010.

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CONTENTS

INTRODUCTION

INTRODUCTION

Acute myocardial infarction (AMI) is the rapid development of myocardial necrosis caused by a critical imbalance between oxygen supply and demand of the myocardium. Myocardial infarction continues to be a significant problem in developed countries and is becoming an increasingly significant problem in developing countries. [1]

Indians develop Coronary Artery Disease (CAD) 5 to 10 years earlier than in other populations, and the occurence of first myocardial infarction before the age of 40 years is 5 to 10 folds higher.[2] Even among Indians, the prevalence of CAD is not homogeneous and is two fold higher in south indians. [3]

Cigarette smoking has been the single factor very strongly associated with CAD in the young adult[4], Perhaps of equal interest is the lack of a strong association of other traditional risk factors to CAD in the young. Hypertension and lack of exercise are both firmly established risk factors for CAD in general, but they appear to contribute only marginally in this population. [5] Numerous studies [6–9] have demonstrated the surprisingly good prognosis up to three years after diagnosis of CAD in the young adult.

In this study the risk factors, clinical presentation and short term outcome of myocardial infarction in patients less than 40 years has been studied and compared with the older patients.

AIM OF THE STUDY

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1. To evaluate the modifiable and non-modifiable risk factors of acute myocardial infarction in young (< 40 years).
2. To study the clinical profile of acute myocardial infarction in young.
3. To study the short term outcome of acute myocardial infarction in young.
4. To compare the young (< 40 years) with older patients (> 40 years) with reference to the risk factor, clinical profile, and short term outcome.

REVIEW OF LITERATURE

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The conceptual basis for considering specific "cardiovascular risk factors" only emerged when the initial findings of the Framingham Heart Study began to appear in the early 1960s. A risk factor is a characteristic or feature of an individual or population that is present early in life and is associated with an increased risk of developing future disease.

The risk factors are primarily considered under the following two categories

Conventional risk factors: [10]

Smoking

Hypertension

Diabetes mellitus

Hyperlipidemia and Elevated Low-Density Lipoprotein Cholesterol

High-Density Lipoprotein Cholesterol

Triglyceride-Rich Lipoproteins

Metabolic Syndrome, Insulin Resistance and Diabetes

Obesity

Family history

Mental Stress, Depression, and Cardiovascular Risk

Novel atherosclerotic risk factors:

High-Sensitivity C - Reactive Protein

Other Markers of Inflammation

Homocysteine

Fibrinogen and Fibrin D-Dimer

Markers of Fibrinolytic Function

Lipoprotein (a)

SMOKING:

Cigarette smoking has been the single factor most strongly associated with CAD in the young adult. A strong dose response relationship between cigarette smoking and CAD has been observed in both sexes, in the young and in the elderly. More than 1 in every 10 cardiovascular deaths in the world in the year 2000 was attributable to smoking [(11)].

It causes acute unfavorable effects on blood pressure and sympathetic tone, and a reduction in myocardial oxygen supply, enhanced oxidation of low-density lipoprotein (LDL) cholesterol and impaired endothelium-dependent coronary artery vasodilation. [(12)]

It also increases levels of CRP, soluble intercellular adhesion molecule-1 (ICAM-1), fibrinogen, and homocysteine[(13)], spontaneous platelet aggregation, increased monocyte adhesion to endothelial cells, and

adverse alterations in endothelial derived fibrinolytic and antithrombotic factors, including tissue-type plasminogen activator and tissue pathway factor inhibitor.

Compared with nonsmokers, smokers have an increased prevalence of coronary spasm and reduced thresholds for ventricular arrhythmia. Even among non-smokers, passive exposure increases the incidence of coronary artey disease. Studies suggest that compared to non- smokers those who consume 20 or more cigarettes per day have a twofold or threefold increased risk of coronary artery disease. (14)

Smoking in the presence of other coronary risk factors appear to have synergistic effect on cardiovascular disease morbidity and mortality. (15)

HYPERLIPIDEMIA:

Abnormalities in plasma lipoproteins and derangements in lipid metabolism rank among the most firmly established and best understood risk factors for atherosclerosis. Atherosclerotic involvement of coronary arteries is well established in men by young adult hood.(16)

Current ATP III guidelines recommend lipid screening in all adults >20 years. ATP III guidelines strive to match the intensity of treatment to an

individual's risk. A quantitative estimate of risk places individuals in one of three treatment strata.

The first step in applying these guidelines involves counting an Individual's risk factors. Individuals with fewer than two risk factors fall into the lowest treatment intensity stratum [LDL goal<160 mg/dL]. In those with two or more risk factors, the next step involves a simple calculation that estimates the 10-year risk of developing coronary heart disease (CHD) Those with a 10-year risk <20% fall into the intermediate stratum [LDL goal <130 mg/dL].

Those with a 10-year CHD risk of >20%, any evidence of established atherosclerosis, or diabetes (now considered a CHD risk-equivalent) fall into the most intensive treatment group [LDL <100 mg/dL]. Members of the ATP III panel have recently suggested 70 mg/dL as a goal for very high risk and as an optional goal for high-risk patients based on recent clinical trial data. [17]

Both hyperlipidemia and hypertriglyceridemia are important risk factors for coronary artery disease. A 10 percent increase in the serum cholesterol result in 20 –30 % increase in the risk of coronary artery disease. if it occurs early in life it may be associated with higher increase in risk. [11]

The risk of CAD is positively correlated with serum total cholesterol concentration, which in turn is highly correlated with LDL levels. LDL has been found to be pro-inflammatory, elevated LDL appears to be involved with all stages of atherogenesis.

Endothelial dysfunction, plaque formation and growth, plaque instability and disruption and thrombosis, Elevated levels of LDL in the plasma leads to increased retention of LDL particles in the arterial wall, their oxidation and secretion of various inflammatory mediators and chemo attractants.

Although LDL is the primary lipid risk factor, other lipids increase the risk of CAD with or without LDL.

The combination of elevated concentrations of triglycerides, small, dense LDL and low levels of HDL is referred to as Atherogenic dyslipidemia. Meta analysis of multiple prospective studies, strongly suggest that elevated serum triglycerides are an independent risk factor for CAD. [18]

Two important mechanisms by which HDL is thought to play a protective role against atherosclerosis are reverse cholesterol transport and inhibition of LDL oxidation. [11] HDL could ferry cholesterol from the vessel wall, augmenting peripheral catabolism of cholesterol. HDL can also carry

antioxidant enzymes that may reduce the levels of oxidized phospholipids in atheromatous lesions, which might enhance atherogenesis. Each increase of HDL cholesterol by 1 mg/dl is associated with a 2 to 3 percent decrease in risk of total cardiovascular disease.

Isser et al. [19] found significant elevation of triglycerides and depression of high-density lipoprotein (HDL) cholesterol in young patients presenting with MI.

HYPERTENSION:

Major overviews and randomized trials continue to demonstrate that blood pressure reductions as small as 4 to 5 mmHg result in large and clinically significant reductions in risk coronary heart disease in middle-aged subjects, the elderly, and high-risk groups, such as those with diabetes and peripheral arterial disease.[20,21]

Both systolic and diastolic hypertension have a strong positive, continuous and graded relationship to CAD.

The potential mechanisms by which hypertension causes coronary events include impaired endothelial function, increased permeability to lipoproteins, increased adherence to leucocytes, increased oxidative stress,

hemodynamic stress triggering plaque rupture and increased myocardial wall stress and oxygen demand. [2]

The JNC VII report has also suggested a new classification of blood pressure, with normal defined as less than 120 mmHg systolic and less than 80 mmHg diastolic.

"Prehypertension" defined as systolic blood pressure 120 to 139 mmHg or diastolic blood pressure 80 to 89 mmHg. Stage 1 hypertension (systolic blood pressure 140 to 159 mmHg or diastolic blood pressure 90 to 99 mmHg) or stage 2 hypertension (systolic blood pressure higher than 160 mmHg or diastolic blood pressure higher than 100 mmHg).[17]

DIABETES:

Diabetes is diagnosed if the fasting blood sugar is > 126 mgs/dl or 2 – hour plasma glucose more than 200mgs/dl. [22]

Diabetes (both type I and II) increases the risk of CAD by 2- 4 times. Approximately 25% of MI survivors have diabetes. Once a type II DM patient suffer an MI, their prognosis for recurrent MI and survival is much worse than that of CAD without DM. Diabetes absolutes the usual protection from CHD afforded for a premenopausal women. Diabetic women have twice the risk of recurrent MI compared to diabetic men.

Mechanisms by which DM causes atherosclerosis includes low HDL-C, high triglycerides, increased lipoprotein remnant particles, increased small dense LDL-C, elevated lipoprotein A concentration, enhanced lipoprotein oxidation, glycation of LDL-C, increased fibrinogen, increased platelet aggregability, increased CRP, PAI-I, impaired fibrinolysis, increased VWF, hyperinsulinemia.[11]

The abnormal lipoprotein profile associated with insulin resistance, known as diabetic dyslipidemia, accounts for part of the elevated cardiovascular risk in patients with type 2 diabetes. While diabetic patients often have LDL cholesterol levels near average, the LDL particles tend to be smaller and denser, and, therefore, more atherogenic. Other features of diabetic dyslipidemia include low HDL and elevated triglyceride levels. [17]

The in- hospital mortality from MI in patients with diabetes is 1.5 to 2 fold higher than non- diabetics. The worse clinical outcome of patients with DM is the acute phase of infarction include

1. The diffuse nature of coronary atherosclerosis
2. Reduced ability to develop collateral blood flow
3. An abnormal pattern of exogenous substrate use in the setting of ischemia leading to increased oxygen consumption by myocardium. [23]

METABOLIC SYNDROME:

The ATP III guidelines now recognize this cluster of risk factors and provide criteria for diagnosis of the "metabolic syndrome"[24]

Abdominal obesity

a. Men (waist circumference) > 102 cm (>40 in.)

b.Women > 88 cm (>35 in.)

Triglycerides >1.7 mmol/L (>150 mg/dL)

HDL cholesterol

a.Men <1.0 mmol/L (<40 mg/dL)

b.Women <1.3 mmol/L (<50 mg/dL)

Blood pressure 130/85 mmHg

Fasting glucose >6.1 mmol/L (>110 mg/dL).

Accelerated and premature CAD in Indians is attributable to the metabolic syndrome [25]

However, not all those with metabolic syndrome have similar risk. Most importantly, endothelial inflammation is now recognized as a major component of metabolic syndrome [26] and the most recent definition of metabolic syndrome from the National Heart, Lung, and Blood Institute

includes a pro-inflammatory state. [27] Thus, inflammatory biomarkers such as hsCRP may help further stratify clinical risk and improve the prognostic value of metabolic syndrome.

OBESITY:

Obesity accelerates the progression of coronary atherosclerosis in adolescent and young adult men. [28]

Obesity is associated with insulin resistance, hypertension, type 2 DM, hyperinsulinemia, low HDL, hypertriglyceridemia, small dense, LDL, raised CRP, thrombosis , diastolic dysfunction and left ventricular hypertrophy [11]

Three key anthropometric measurements are important to evaluate the degree of Obesity — weight, height, and waist circumference.

Excess abdominal fat, assessed by measurement of waist circumference or waist-to-hip ratio, is independently associated with higher risk for diabetes mellitus and cardiovascular disease

South Asians and Chinese	
Men	>90 cm (35 in)

Women	>80 cm (31.5 in)

The body mass index (BMI), calculated as weight (kg)/height $(m)^2$

	BMI kg/ m^2	Obesity Class	Risk of Disease
underweight	<18.5		
Healthy weight	18.5-24.9		
Overweight	25.-29.9		Increased
Obesity	30.0-34.9	I	High
Obesity	35.0–39.9	II	Very high
Extreme obesity	>40	III	Extremely high

FAMILY HISTORY:

A family history of premature atherosclerosis, increases the risk of atherosclerosis in an individual likely as a result of environmental factors and a genetic predisposition to the disease.[29]

A growing body of evidence, for example, shows that specific lipid abnormalities may be genetically transmitted to the offspring of patients with severe CAD. Uiterwaal et al. [30] found lower levels of HDL_3 cholesterol and apolipoprotein A2 in male children of CAD patients. Similar but more modest trends were also identified in the female children of these patients.

SOCIOECONOMIC STATUS:

Persons with low socioeconomic status are at high risk for CAD. Risk factors for atherosclerosis as smoking, hypertension, obesity and sedentary lifestyle are high in this economic group.

Some of these risk factors as well as the psychomotor stressors increase the exposure to CAD. They also have less access to care. [11]

POST MENOPAUSAL STATE:

After menopause, coronary risk accelerates in women. At least part of the apparent protection against CHD in premenopausal women derives from their relatively higher HDL levels compared with those of men.

After menopause, HDL values fall in concert with increased coronary risk. Estrogen therapy lowers LDL cholesterol and raises HDL cholesterol, changes that should decrease coronary risk.

Young women with CAD comprise an especially interesting group given the protective effect of estrogen, but which factors are predictive in this distinctly unusual cohort is poorly understood.[31] Anecdotal cases suggest that diabetes in women may have a more powerful role than in men. Women who smoke have a quantitatively similar risk as men, [32] but more than five times the risk of nonsmoking women.[33]

Smoking in combination with oral contraceptives poses a 13-fold increase in CAD mortality.[34] Truncal obesity and increased body mass index (BMI) have recently been proposed as potential independent risk factors, particularly in young women with CAD.

NOVEL ATHEROSCLEROTIC RISK FACTORS:

Although the use of global prediction models like those developed in Framingham greatly improves the detection of heart disease risk, as many as 20 percent of all events occur in the absence of any of the major classic vascular risk factors. Thus, because of the considerable need to improve vascular risk detection, much research over the past 10 or 15 years has focused on the identification and evaluation of novel atherosclerotic risk factors. [35]

HIGH-SENSITIVITY C-REACTIVE PROTEIN

The acute-phase reactant, CRP, a simple downstream marker of inflammation, has now emerged as a major cardiovascular risk factor.[36] Composed of five 23-kDa subunits, CRP is a circulating member of the pentraxin family that plays a major role in the human innate immune response

More than simply a marker of inflammation, CRP may influence directly vascular vulnerability through several mechanisms, including enhanced expression of local adhesion molecules, increased expression of endothelial PAI-1, reduced endothelial nitric oxide bioactivity, altered LDL uptake by macrophages, and colocalization with complement within

atherosclerotic lesions. Moreover, expression of human CRP in CRP-transgenic mice was found to directly enhance intravascular thrombosis.[37]

hsCRP levels less than 1, 1 to 3, and higher than 3 mg/liter should be interpreted as lower, moderate, and higher relative vascular risk, respectively, when considered along with traditional markers of risk.[38, 39,40, 41].

hsCRP adds prognostic information at all levels of LDL cholesterol and at all levels of risk, as determined by the Framingham Risk Score. In other major studies, hsCRP levels predicted subsequent risk better than LDL cholesterol level.

OTHER MARKERS OF INFLAMMATION : [10]

Although hsCRP is by far the best-characterized and most reliable inflammatory biomarker for clinical use, several other markers of inflammation have shown promise in terms of predicting vascular risk.

These include cytokines such as interleukin-6, soluble forms of certain cell adhesion molecules such as intercellular adhesion molecule (sICAM-1), P-selectin, or the mediator CD40 ligand, as well as markers of leukocyte activation such as myeloperoxidase. Other inflammatory markers

associated with lipid oxidation such as lipoprotein-associated phospholipase A_2 and pregnancy-associated plasma protein A have also shown promise

HOMOCYSTEINE: [10]

Homocysteine is a sulfhydryl-containing amino acid derived from the demethylation of dietary methionine. Patients with rare inherited defects of methionine metabolism can develop severe hyperhomocysteinemia (plasma levels higher than 100μmol/liter) and have markedly elevated risk of premature atherothrombosis as well as venous thromboembolism.

Mechanisms suggested to account for these effects include endothelial dysfunction, accelerated oxidation of LDL cholesterol, impairment of flow-mediated endothelium-derived relaxing factor with subsequent reduction in arterial vasodilation, platelet activation, and oxidative stress.

In contrast to severe hyperhomocysteinemia, mild to moderate elevations of homocysteine (plasma levels higher than 15μmol/liter) are more common in the general population, primarily because of insufficient dietary intake of folic acid. Despite reduced enthusiasm and lack of evidence that homocysteine reduction lowers risk, there remain a few specific patient populations for whom homocysteine evaluation may prove appropriate, including those lacking traditional risk factors, with renal failure, or with

markedly premature atherosclerosis or a family history of myocardial infarction and stroke at a young age.[42]

FIBRINOGEN AND FIBRIN D DIMER:

Plasma fibrinogen influences platelet aggregation and blood viscosity, interacts with plasminogen binding and, in combination with thrombin, mediates the final step in clot formation and the response to vascular injury

Fibrin D-dimer reflects the extent of fibrin turnover in the circulation, and epidemiological evidence has found D-dimer levels to have modest predictive value for future vascular events.

Following myocardial infarction, D-dimer levels also predict recurrent events and, like CRP, D-dimer predicts poor outcome in troponin-negative ischemia.[43]

MARKERS OF FIBRINOLYTIC FUNCTION: [10]

Impaired fibrinolysis can result from an imbalance between the clot-dissolving enzymes t-PA or urokinase-type plasminogen activator and their endogenous inhibitors, primarily PAI-1. Plasma levels of PAI-1 peak in the morning, whereas concentrations of t-PA demonstrate a less prominent circadian variation.

On this basis, a relative hypofibrinolytic state may prevail in the morning that, along with increased platelet reactivity, may contribute to the increased risk of myocardial infarction seen during this period. The clinical use of fibrinolytic markers to determine coronary risk offers only marginal value, and no data available suggest that measures of fibrinolysis add to traditional risk scores.

LIPOPROTEIN (a): [10]

Lipoprotein(a) (Lp[a]) consists of an LDL particle with its apolipoprotein B-100 (apo B-100) component linked by a disulfide bridge to apolipoprotein(a) (apo[a]), The close homology between Lp(a) and plasminogen has raised the possibility that this lipoprotein may inhibit endogenous fibrinolysis by competing with plasminogen binding on the endothelium. More recent studies have suggested that Lp(a) binds and inactivates tissue factor pathway inhibitor and may upregulate the expression of plasminogen activator inhibitor, further linking lipoproteins and thrombosis.

Lp(a) also colocalizes within atherosclerotic lesions and may have local actions through oxidized phospholipids pathways.(44) Thus, several mechanisms may contribute to a role of Lp(a) in atherothrombosis.

Recent data [45] suggest that elevated homocysteine and elevated Lp(a) are independent risk factors for the development of CAD in young men; however, these two factors appear to interact in a synergistic fashion to confer risk in young women

OTHER FACTORS; [10,11]

Physical exercise reduces myocardial oxygen demand and increases exercise capacity, both of which correlate with lower levels of coronary risk. The cardioprotective effects of exercise include reduced adiposity and diabetes incidence, lowered blood pressure, and improvement of dyslipidemia, as well as vascular inflammation. Exercise also enhances endothelial dysfunction, insulin sensitivity, and endogenous fibrinolysis.

MENTAL STRESS:

Entirely unexplored areas like anger and psychosocial stress can add a significant morbidity and were associated with MI and coronary artery calcification in young adults. [46]

Both depression and mental stress predispose to increased vascular risk. The adrenergic stimulation of mental stress can augment myocardial oxygen requirements and aggravate myocardial ischemia. Mental stress can cause coronary vasoconstriction, particularly in atherosclerotic coronary

arteries, and hence can influence myocardial oxygen supply as well. Studies have further linked mental stress to platelet and endothelial dysfunction, the metabolic syndrome, and the induction of ventricular arrhythmias. [10]

Congenital coronary artery anomalies can present for the first time as MI in young adults. Cases of myocardial bridging, where the coronary arteries are embedded within a tunnel in the myocardium beneath a layer of muscles, have also been reported in young people presenting with MI. Myocardial bridging can result in significant ischaemia during systolic contraction and can result in MI. [47 – 49]

CLINICAL PRESENTATION:

Chest pain is the commonest presenting symptom. The pain is prolonged, usually lasting for more than 30 minutes and frequently for a number of hours. The discomfort is described as constricting, crushing, oppressing, or compressing; often the patient complains of a sensation of a heavy weight or a squeezing in the chest.

The classic presentation of worsening angina culminating in MI is rare in younger patients. The first onset of angina that rapidly progresses to fully evolved MI is often the case in younger patients. [50]

Atypical presentations of AMI include the following:

1. Heart failure (i.e., dyspnea without pain beginning de novo or worsening of established failure);
2. Classic angina pectoris without a particularly severe or prolonged episode;
3. Atypical location of the pain;
4. Central nervous system manifestations, resembling those of stroke, secondary to a sharp reduction in cardiac output in a patient with cerebral arteriosclerosis;
5. Apprehension and nervousness;
6. Sudden mania or psychosis;
7. Syncope;
8. Overwhelming weakness;
9. Acute indigestion; and
10. Peripheral embolization.

Approximately one fourth of patients, MI is associated with no symptoms at all. [51]

PHYSICAL EXAMINATION:

The physical examination can often be unremarkable. [10, 11, 17]

- Patients with ongoing symptoms usually lie quietly in bed and appear pale and diaphoretic.

- Hypertension may precipitate MI, or it may reflect elevated catecholamine levels due to anxiety, pain, or exogenous sympathomimetics.
- Hypotension may indicate ventricular dysfunction due to ischemia. Hypotension in the setting of MI usually indicates a large infarct secondary to either decreased global cardiac contractility or a right ventricular infarct.
- Acute valvular dysfunction may be present. Valvular dysfunction usually results from infarction that involves the papillary muscle. Mitral regurgitation due to papillary muscle ischemia or necrosis may be present.
- Rales may represent congestive heart failure.
- Neck vein distention may represent pump failure. With right ventricular failure, cannon jugular venous *a* waves may be noted.
- Third heart sound (S_3) may be present.
- A fourth heart sound is a common finding in patients with poor ventricular compliance that is due to preexisting heart disease or hypertension.
- Dysrhythmias may present as an irregular heartbeat or pulse.
- Low-grade fever is not uncommon

SITE OF MI:

The site of myocardial infarction depending on the vessels occluded.

Left anterior descending (LAD)	- Antroseptal MI
Left circumflex artery	- Anterolateral MI
Right coronary artery	- Inferoposterior wall MI With or without RVMI

The site of MI based on the ECG findings. [52]

1. Extensive Anterior wall MI : ST segment elevation in leads I, AVL and anterior precordial leads .

2. Anteroseptal MI : ST segment elevation in leads v1 to v4.

3. Anterolateral wall MI : ST segment elevation in leads I, AVL, V4 to V6.

4. Inferior wall MI : ST segment elevation in leads II, III, AVF.

5. Inferolateral MI: ST segment elevation in leads II, III, AVF and V5 to V6.

6. Posterior wall MI: the following findings in leads V1 to V3.

- The mirror image of QS complex reflected by a all and widened R wave
- The mirror image of ST segment elevation reflected by a widened ST segment depression.
- The mirror image of inverted T wave reflected by a tall, widened and upright T wave.

7. Right ventricular MI: in the presence of inferior wall MI, ST segment elevation of 1mm or more in V1 ,V4R to V6R.

MATERIALS AND METHODS

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POPULATION

Eighty cases of acute myocardial infarction were chosen from the Intensive Coronary Care Unit at the Government Royapettah Hospital, Chennai from January 2009 – October 2009 for the study. Forty cases of age less than 40 years (Group I)and forty patients of age more than 40 years (Group II) were taken and the observations was made.

INCLUSION CRITERIA

1. ECG proven acute myocardial infarction.

2. Patients who underwent thrombolytic therapy.

EXCLUSION CRITERIA

1. Any contraindication to thrombolytic therapy.
2. Any myocardial infarction in past.

CONTRAINDICATION TO THROMBOLYTIC THERAPY [10]

Absolute Contraindication

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (eg- arteriovenous malformation)
- Known malignant intracranial neoplasms (primary or metastatic)
- Ischemic stroke within 3 months (except acute ischemic stroke within 3 hr)
- Suspected Aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 months

Relative contraindications

- History of chronic severe poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP > 180mmHg or DBP > 110mmHg)
- History of prior ischemic stroke > 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged CPR (> 10min) or major surgery with in 3wks
- Recent (within 2 – 4 wk) internal bleeding
- Non-compressible vascular punctures
- For streptokinase : prior exposure > 5 days ago or prior allergic reaction to the drug
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR the risk of bleeding.
- A detailed history especially with reference to the presence of risk factors like diabetes mellitus, hypertension, smoking, obesity, family history, hypercholesterolemia, and previous history of coronary

artery disease was taken. Age, sex, marital status, occupation, religion and time window were also noted. At the time of admission the pulse rate, blood pressure, Killip's class, was noted and a detailed physical examination was done. Laboratory parameters done were complete hemogram, blood sugar (fasting and post prandial), blood urea, serum creatinine, serum electrolytes, serum cholesterol, bleeding time and clotting time. ECG at the time of admission and after 6 hrs was taken and assessed for ST elevation and arrhythmias. Chest X rays were also taken.

KILLIPS CLASS

Class I : no signs of pulmonary venous congestion

Class II : moderate heart failure, as evidenced by rales over less than 50% of lung, S_3 gallop, tachypnoea, or signs of failure of the right side of the heart including hepatic and venous congestion

Class III: severe heart failure, pulmonary edema (with rales over more than 50% of the lungs).

Class IV: shock with systolic blood pressure less than 90 mmHg and evidence of peripheral constriction, diaphoresis, peripheral cyanosis, mental confusion and oliguria.

All patients were given injection pethidine 50mg iv, injection, promethazine (25mg iv), aspirin 150 mg OD, clopidogrel 75 mg OD, oxygen (2 – 4 l/ min) and IV heparin (5000 U 6^{th} hourly for 5- 7 days).

All patients were given injection Streptokinase 1.5 million units dissolved in 100ml of normal saline over 45 – 60 min. Patients who presented with high blood pressure, were treated with nitroglycerin infusion and then thrombolysed.

Subsequently during the following days, all patients in both the groups were started on Tab. Atenolol 50 mg once daily and Tab. Enalapril 2.5 mg once daily, provided there were no contraindications to therapy.

All these patients were intensively followed up for development of lung signs, bleeding complications, arrhythmias, hypotension, post infarction angina, reinfarcts and use of antiarrhythmic and other drugs. All patients underwent echocardiogram on day 6, prior to discharge.

Echocardiography was done by HP Image Point machine with 2D Echo, M mode and Color Doppler study was done in all patients using parasternal and apical windows. The views studied were parasternal long axis, parasternal short axis, apical 4 chamberview and apical 2 chamber views. Features noted were 2D dimension of interventricular septum, left

ventricular free wall, thickening of the walls, fractional shortening of Inter ventricular(IV) septum and free wall, studies of the valves, presence of hypokinesia, akinesia or dyskinesia of IV septum and free wall. All patients were screened for presence of effusion, thrombus, calcification and vegetation. Calculations obtained were LV diastolic volume, LV systolic volume, fractional shortening and ejection fraction.

Doppler study was used to measure LV inflow pattern, E/A ratio (which denotes flow velocities across the mitral valve) to note the presence of diastolic dysfunction.

Both age groups were compared for sex, occupation, time window, site of infarct, risk factors (diabetes mellitus, hypertension, smoking, obesity, family history, hypercholesterolemia, and previous history of coronary artery disease), the pulse rate, blood pressure, Killip's class, ECG parameters, hemoglobin, blood sugar, serum cholesterol, LDL, HDL, TAG, arrhythmia, signs of failure, post infarct echo.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

SEX

The patients were stratified into males and females in both group I (< 40 years) and group II (> 40 years)

Table: 1

	GROUP I	GROUP II
MALE	38	28
FEMALE	02	12

There were two females in group I, whereas twelve females in group II.

There is significant difference in the two groups (P value 0. 003).

OCCUPATION: Table 2

Occupation	Group I	Group II
Professionals and officials	3	3
Clerical	6	6
Manual laborers	25	16
House wife	1	10
Others	5	5

Occupation wise MI was commonly seen in the manual labourers in both the groups, but the difference in two groups is not statistically significant. (P= 0.053)

SMOKING:

Table: 3

Smoking in cigarette pack years	Group I	Group II
0	7	21
<10	6	3
11 – 20	11	5
21 – 30	10	5
31 – 40	5	4
> 40	1	2

(1 cigarette pack year = 20 cigarette/ day for 1 year)

33 patients in the group I and 19 patients in the group II were smokers.

7 patients were non smokers in group I and 21 patients were non smokers in group II. There is statistically significant difference in the two groups. (P = 0.030)

BMI:

Table: 4

	GROUP I	GROUP II
NORMAL	01	00
OVER WEIGHT	10	05

OBESITY I	10	07
OBESITY II	17	24
OBESITY III	02	04

There is no statistically significant difference in BMI between the groups. (p = 0.281)

SITE OF INFARCT **Table 5**

Site		GroupI	Group II
Inferior	IWMI	12	12
	ILMI	6	2
	IPL	2	5
Anterior	AWMI	17	17
	EAMI	3	4

Inferior wall MI was 12 in both the groups. Inferolateral MI was 6 in group I and 2 in group II. Inferoposterolateral MI was 2 in group I and 5 in group II. Anterior wall MI was 17 in both the groups. Extensive Anterior wall MI was 3 in group I and 4 in group II. The difference is not statistically significant.

DIABETES MELLITUS:

Table: 6

Diabetes mellitus	Group I	Group II
Positive	5	20
Negative	25	20

In group I 5 patients were diabetic and in group II 20 were diabetic. The difference is statistically significant. (P < 0.001)

HYPERTENSION: **Table: 7**

Hypertension	Group I	Group II
Positive	7	17
Negative	33	23

In group I, 7 patients were hypertensives and in group II, 17 were hypertensives. The difference is statistically significant. (P = 0.015)

FAMILY HISTORY:

Table: 8

Family History of IHD	Group I	Group II
Positive	25	11
Negative	15	29

Twenty five patients in group I and eleven patients in the group II had Positive family history of IHD. This is statistically significant (P = 0.002).

TIME WINDOW:

Mean time window in Group I was 3.58 ± 1.80hrs. Mean time window in Group II was 3.22 ± 1.62 hrs. The difference is not statistically significant. (P =0.349).

PULSE RATE:

The mean pulse rate in Group I was 85.2 ± 14.87/min and

Mean pulse rate in Group II was 90.05 ± 15.29 /min. There is no significant Difference in the two groups (P = 0.161).

SYSTOLIC BLOOD PRESSURE:

Table 9

	Group I	Group II
Normal	19	10
Pre hypertension	12	8
Stage I	5	16
Stage II	4	6

Nineteen patients had normal systolic blood pressure in Group I and ten Patients had normal systolic blood pressure in Group II. The difference is

Statistically significant. (P= 0.021)

DIASTOLIC BLOOD PRESSURE:

Table 10

	Group I	Group II
Normal	19	10
Pre hypertension	15	8
Stage I	2	16
Stage II	4	6

Nineteen patients had normal diastolic blood pressure in Group I and ten Patients had normal diastolic blood pressure in Group II. The difference isStatistically significant (P= 0.001).

Killip's Class:

Table 11

	GROUP I	**GROUP II**
CLASS I	**35**	**30**
CLASS II	**3**	**7**
CLASS III	**1**	**2**
CLASS IV	**1**	**1**

Thirty five patients in Group I and thirty patients in group II presented with killips class I. the difference is not statistically significant (P=0.509).

ST SEGMENT ELEVATION:

The mean ST elevation in Group I was 7.9 ± 2.95mm and in Group II was 7.57±2.57mm .The difference between the mean is not statistically Significant. (P = 0.601)

FASTING BLOOD SUGAR:

Table 12

FBS	GROUP I	GROUP II
< 125	34	22
>125	6	18

Six patients had high fasting sugar level in Group I and eighteen patients had raised fasting sugar levels in Group II. The difference is statistically Significant. (P=0.003)

POST PRANDIAL BLOOD SUGAR:

Table 13

PPBS	GROUP I	GROUP II
<200	35	22
>200	5	18

Five patients had high post prandial sugar level in Group I and eighteen patients had raised postprandial sugar levels in Group II. The difference is statistically significant. (P=0.001)

TOTAL CHOLESTEROL

Table 14

TC	GROUP I	GROUP II
<200	30	25
>200	10	15

Total cholesterol was high in ten patients in Group I whereas fifteen patients in Group II . The difference is not statistically significant. (P=0.228)

TRIACYL GLYCEROL:

Table 15

TAG	GROUP I	GROUP II
<150	27	22
>150	13	18

Triglycerides was high in thirteen patients in Group I and eighteen patients in Group II .The difference is not statistically significant. (P=0.251)

LOW DENSITY LIPOPROTEIN:

Table 16

	GROUP I	GROUP II
<100	21	15
>100	19	25

Low density lipoproteins was high in nineteen patients in Group I and twenty five patients in Group II .The difference is not statistically Significant. (P=0.178).

HIGH DENSITY LIPOPROTEIN:

Table 17

	GROUP I	GROUP II
Normal	11	16
Reduced	29	24

High density lipoproteins were low in twenty nine patients in Group I whereas in group II it was low in twenty four patients. The difference is not statistically significant. (P=0.178)

EJECTION FRACTION:

Table 18

	GROUP I	GROUP II
LESS THAN 60%	12	29
MORE THAN 60%	38	1

Ejection fraction was less than 60% in 12 patients in Group I and in 29 Patients in Group II. The difference is statistically significant (P<0.001).

WALL MOTION ABNORMALITY

Table 19

	GROUP I	**GROUP II**
PRESENT	**23**	**26**
ABSENT	**17**	**14**

Twenty three patients in Group I and 26 patients in Group II had wall motion abnormality. The difference is not statistically significant. (p=0.491)

REINFARCTION:

Table 20

	GROUP I	GROUP II
ABSENT	39	36
PRESENT	1	4

Reinfarction occurred in one patient in Group I and in four patients in GroupII .The difference is not statistically significant. (P=0.166)

DEATH:

There was one death in group I and three in group II. The difference is not Statistically significant. (P=0.305)

DISCUSSION

DISCUSSION

Premature CAD is a rapidly progressive form of atheromatous process [50] The risk factors and short term outcome of acute myocardial infarction in young adults varied from their elderly counterparts.

Cigarette smoking has been the single factor most strongly associated with CAD in the young adult. Diabetes and hyperlipidemia are also frequently present in young CAD patients.

Whereas the importance of these factors in the pathogenesis of CAD and their powerful relationship to rapid disease progression is well documented [53,54] their importance in this population is not characterized in detail.

In a study done by Kanitz MG et al to define the risk factors and clinical presentation of patients under age 40 who present to the emergency department (ED) of a community hospital with an acute myocardial infarction (AMI), 209 patients were studied. The major risk factor was tobacco use 81%, followed by family history 40%, hypertension 26%, and hyperlipidemia 20%.[55]

In the present study in patients under age 40, tobacco use was present in 82.5%, family history 62.5%, hypertension 52.5%, hyperlipidemia 25%.

In another study conducted by Hong MK et al, 631 patients were studied. Their conclusions were, occurence of Acute myocardial infarction below forty years was the predominant disease of men. Risk factor analysis revealed a history of cigarette smoking and hypercholesterolemia were more frequently found in the young patients, but a history of hypertension and diabetes were more frequently found in the elderly patients. [56]

In this study in less than 40 years group, 95% were men, cigarette smoking was more common in younger patients, whereas hypercholesterolemia was present in both groups. Hypertension and diabetes were more frequent in the elderly group.

In a prospective clinical study conducted by Fournier et al 108 consecutive Mediterranean patients with AMI, aged < 40 years, were prospectively studied. Clinical features, risk factors, and in-hospital morbidity and mortality were evaluated. The most common risk factors were cigarette smoking 94.5% and hypercholesterolemia 48%.and that the short term prognosis was excellent.[57]

The short term outcome in the younger patients in this study was better than the older ones.

Stefanos Garoufalis et al conducted a study of risk factors comparing 460 patients and found that family history and smoking were the most common risk factors in patients ≤45 years old P<0.04, P<0.0001, respectively, and hypertension and diabetes mellitus were more prevalent in patients >45 years, P<0.00001 for both [58].

In this study, we had similar results, with family history (P = 0.002), smoking (P = 0.030) being common in younger patients, and hypertension (P = 0.015) and diabetes (P <0.001).

Colkesen et al surveyed 25,038 patients and studied the risk factors and found that Male sex was more prevalent in acute MI group when compared with control participants (83 vs. 59%, respectively; P=0.01).

A significant difference was found in cigarette smoking (62 vs. 36%, respectively; P=0.007) and family history (33 vs. 16%, respectively; P=0.03) between the two groups. [59]

In another study, 9,373 patients were assessed for the frequency, risk factors, presenting symptoms, treatment, complications and in-hospital outcomes of young patients and compared with those of older patients. .

Risk factors such as tobacco use and a family history were more frequent in the young patients, whereas diabetes and hypertension were less frequent. Importantly, 66% of the patients aged <45 years had a history of tobacco use. Younger patients had a lower in-hospital mortality rate, lower incidence of congestive heart failure and a shorter length of stay. Multivariable analysis of in-hospital mortality revealed that older age remained an independent predictor of death. [60]

SUMMARY

SUMMARY

- There were only 5% females in group I, whereas 30% in group II. The difference was statistically significant (P = 0.003)
- Acute myocardial infarction was more common among the manual labourers in both the groups. There was no significant difference between the groups.
- Smoking was more prevalent in group I compared to group II and the difference was statistically significant(P = 0.030)
- There was more number of diabetics in group II compared to that of group I. The difference was significant (p<0.001)
- Hypertension was less common in group I compared to group II and the difference was significant (P = 0,015)
- There was a significant difference in the distribution of family history between the groups suggesting a higher prevalence of family history of coronary artery disease in less than 40 years.(P = 0.002)

- Although there was no difference in body mass index (BMI) between the two groups, the body mass index was high in both the groups. (P=0.281)

- There was no difference between the groups in terms of site of infarct, pulse rate and time window.
- The systolic and diastolic blood pressure was higher in group II compared to that of group I (P=0.021 and <0.001 respectively)
- There was a no statistically significant difference in the ST segment elevation in the two groups (P = 0.601).
- There was no difference in the killip's class between the two groups.
- Group II had raised fasting and post prandial blood sugar levels compared to group I, with statistically significant P of 0.003 and 0.001 respectively.
- Total cholesterol, triglycerides, low density lipoproteins was elevated in both the groups, but the difference between the groups was not statistically significant.
- HDL was lower than normal in 29 patients in group I and 24 patients in group II, but the difference was not significant.

- There was better ejection fraction in Group I compared to Group II with a significant P value of < 0.001.
- There was no significant difference in reinfarction and death in the two groups.

LIMITATIONS

LIMITATIONS

1. The sample size of forty may be a small number to draw conclusions, however since the study showed significant difference in the risk factors and outcome, a study in a larger group is needed for further validation.

2. Estimation of novel atherosclerotic risk factors could not be done, due to unavailability of the tests .

3. Angiographic correlation could not be done, since the facility was not available .

4. The period of follow up in this study was short term, whereas a long term follow up would give better idea about the outcome of myocardial infarction.

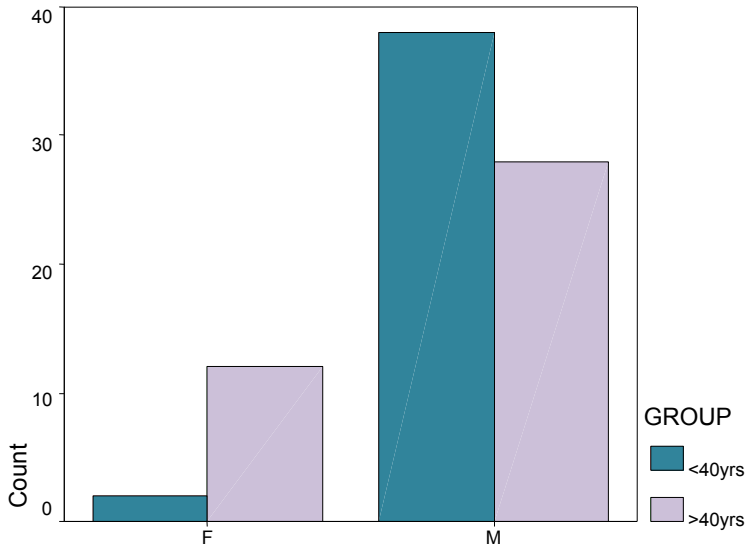
CONCLUSION

CONCLUSIONS

- The frequency of risk factors in young patients differed from those in their elderly counterparts. Smoking and family history were more common risk factors in the younger age group, whereas diabetes and hypertension were common above 40 years.
- There was no difference in the clinical presentation among the two groups.
- The ejection fraction was good in patients less than forty years as compared to the older patients, signifying a better short term outcome in the young patients.
- Though reinfarction and death occurred more frequently in patients more than forty years, the difference was not significant.

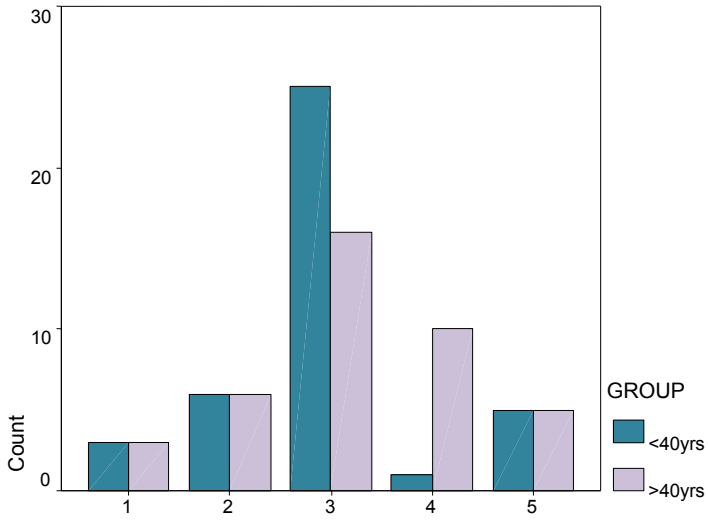
CHARTS

CHART - 1 Sex



SEX

CHART - 2 Occupation



OCC

CHART- 3 Smoking

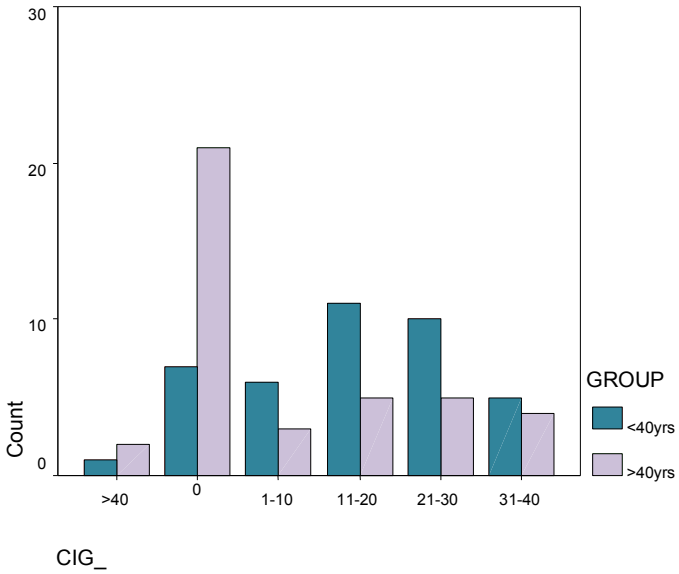


CHART – 4 Body Mass Index

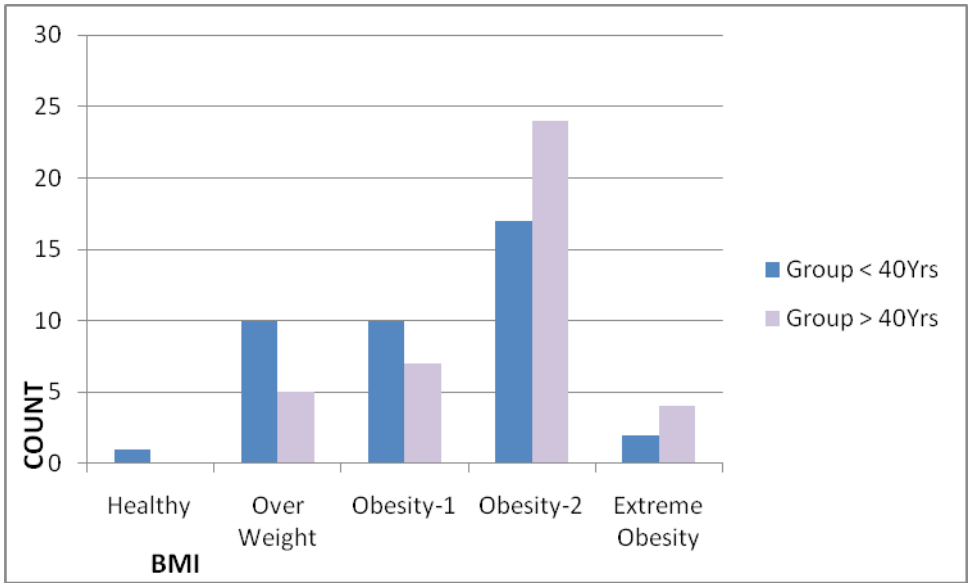
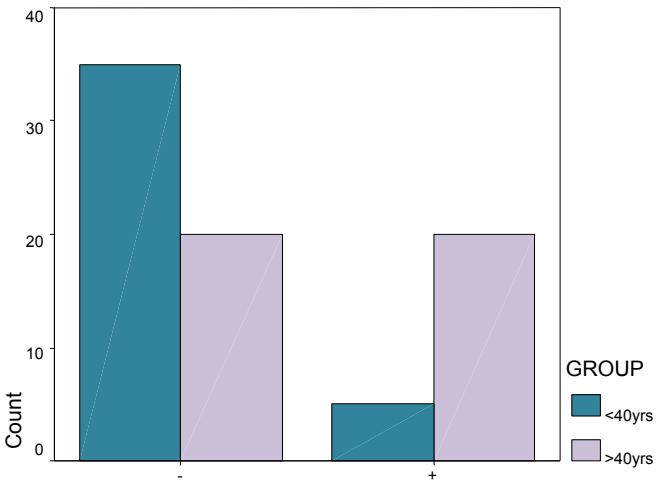
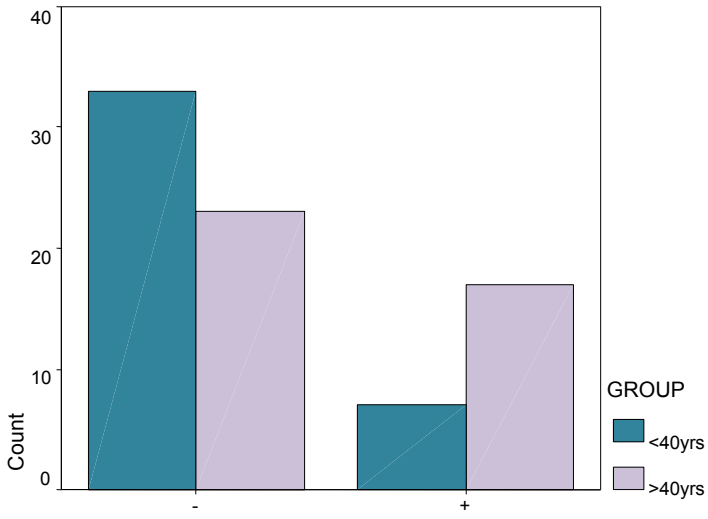


CHART – 5 Diabetes mellitus



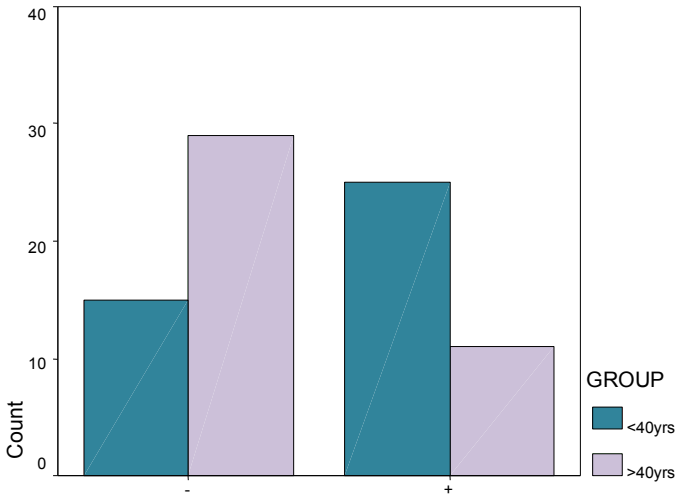
DM

CHART – 6 Systemic hypertension



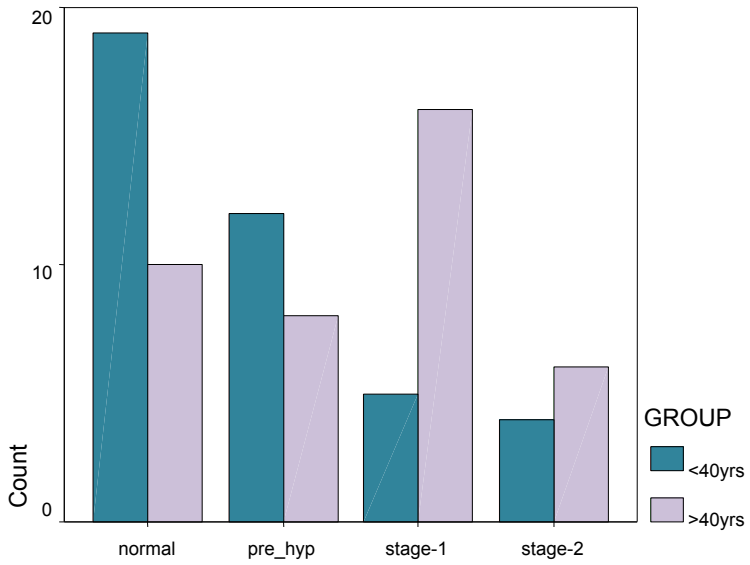
SHT

CHART – 7 Family history



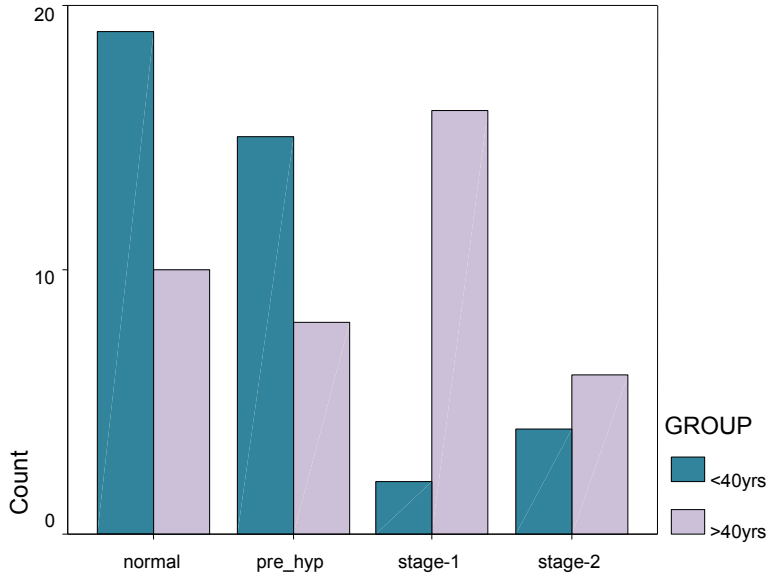
FH

CHART – 8 Systolic blood pressure



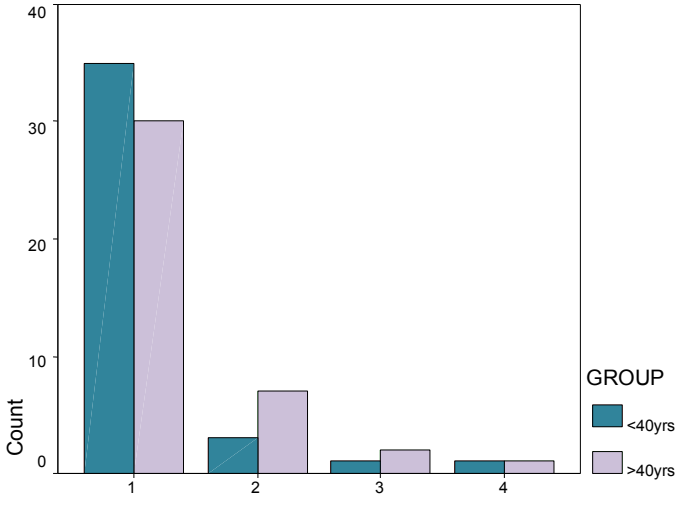
SBP_

CHART – 9 Diastolic blood pressure



DBP__

CHART – 10 Killip's Class



KLP_CLS

CHART - 11 Fasting blood sugar

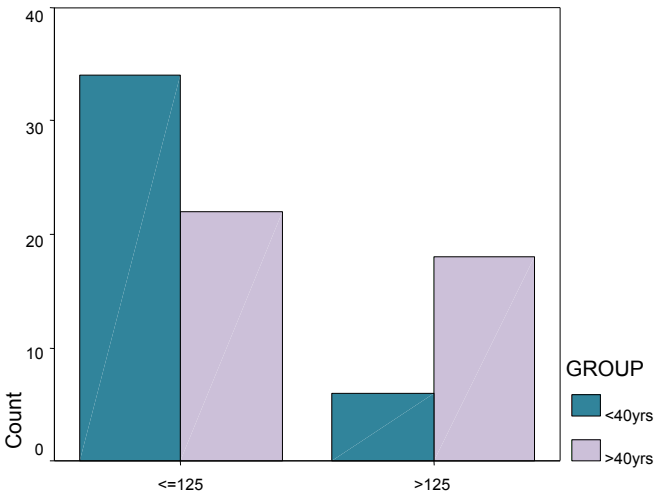


CHART – 12 Post prandial blood sugar

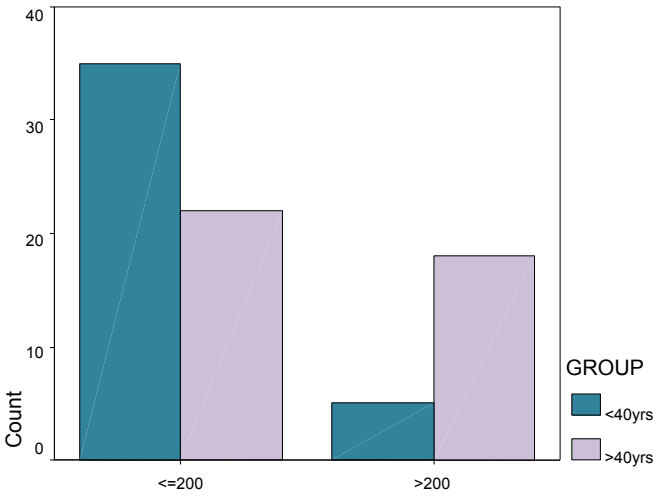


CHART – 13 Total cholesterol

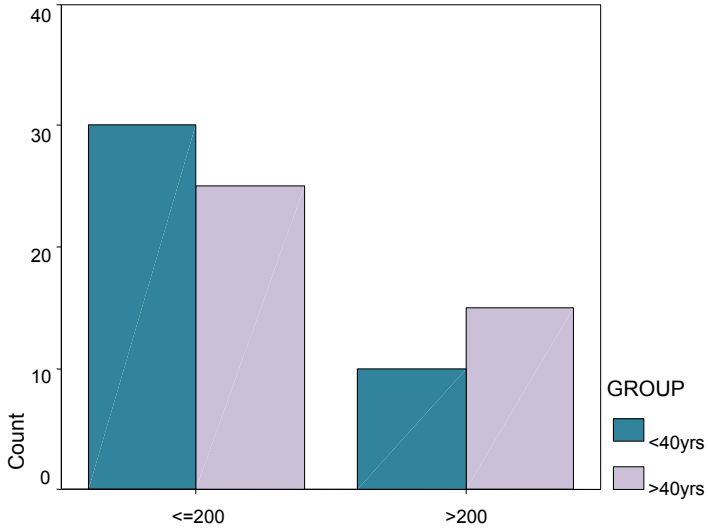


CHART – 14 Triacylglycerol

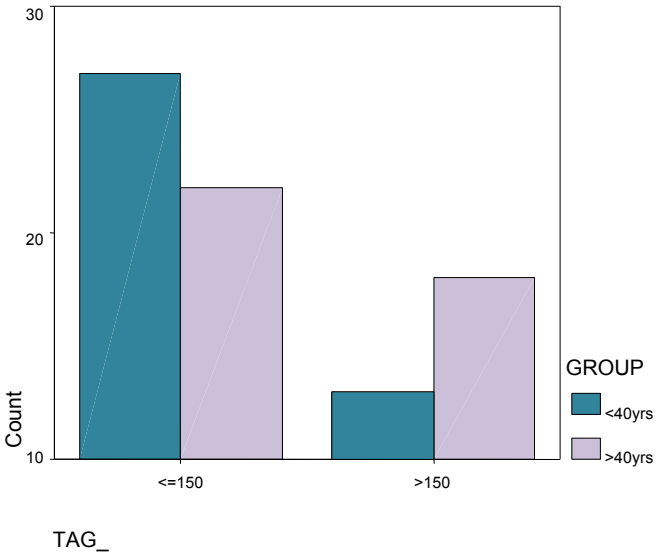
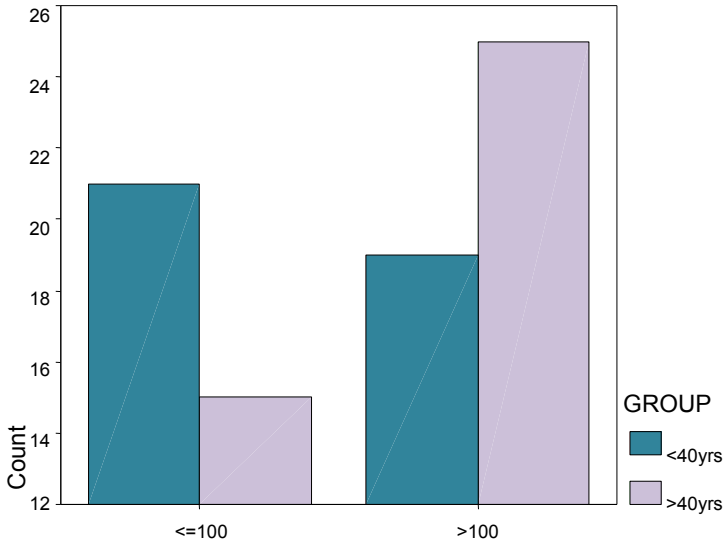
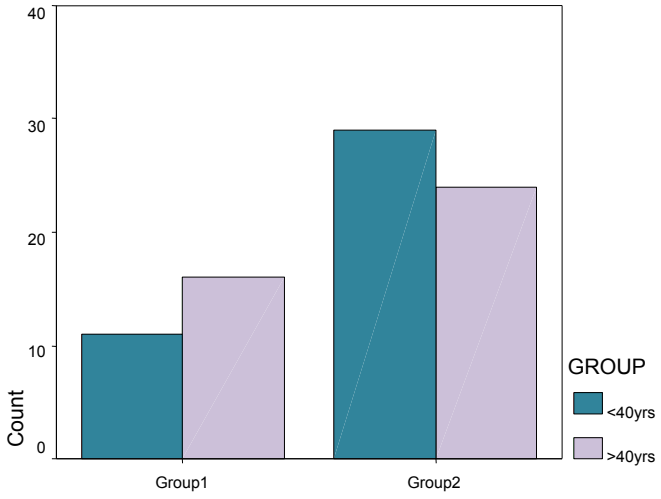


CHART – 15 Low density lipoprotein



LDL_

CHART – 16 High density lipoprotein



HDL_

CHART – 17 Ejection fraction

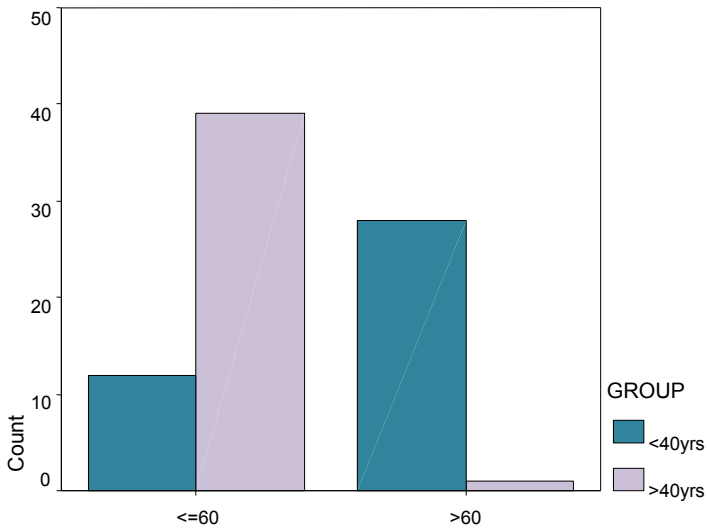


CHART – 18 Wall Motion

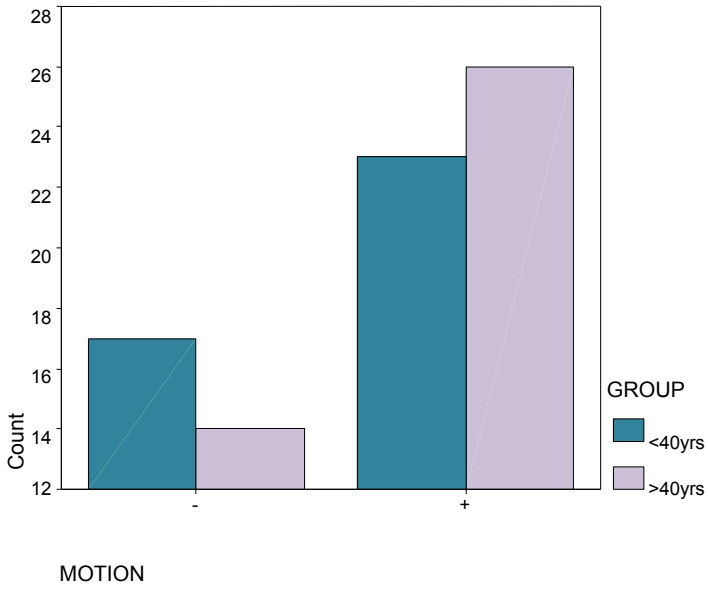
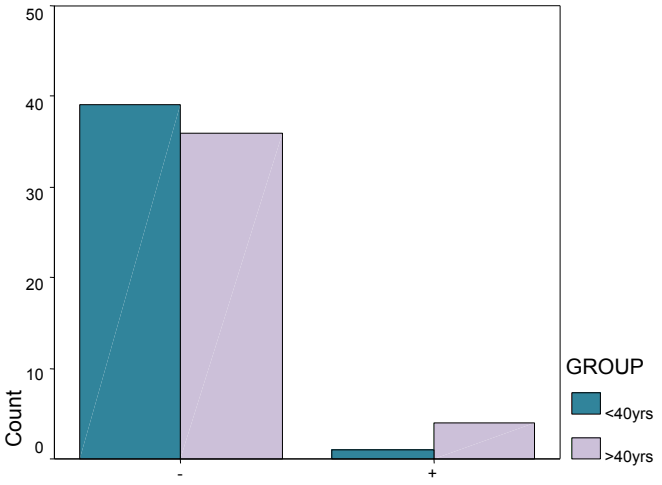
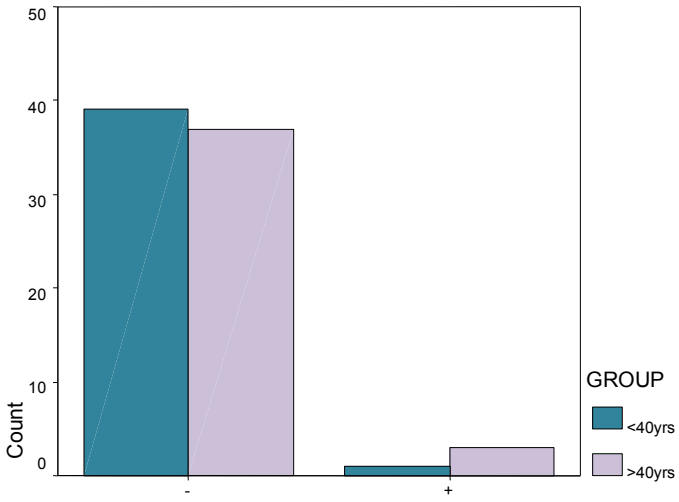


CHART – 19 Reinfarction



RE_INF

CHART – 20 Death



DEATH

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PROFOMA

PROFOMA

1. Name and address : 2. Age yrs 3. Sex:

4. Marital status : 5. Occupation:

6. IP No: 7. DOA: 8. DOD:

Diagnosis:

SYMPTOMS

A.CHEST PAIN - (1) Yes (2) No (3) Non Specific

If yes, is it related to exertion?

(1) Yes (2) No

CLASS - 1,2,3,4

DURATION OF ILLNESS - 1. Less than 6 hours

2. 6 hours to 24 hours

3. More than 24 Hours

B.DYSPNOEA - (1) Yes (2) No

If yes, is it related to exertion?

(1) Yes (2) No

CLASS - 1,2,3,4

DURATION OF ILLNESS - 1. Less than 6 hours

2. 6 hours to 24 hours

3. More than 24 Hours

C.PALPITATION - Yes/NO

D. SWEATING - Yes/NO

E. VOMITING – Yes/No

F. GIDDINESS – Yes/No

G. EXTREME FATIGUE – Yes/No

H. SYNCOPE – Yes/No

I. MISCELLANEOUS –

RISK FACTORS AND PAST HISTORY

1. DIABETES MELLITUS - (1) Yes (2) No (3) Unknown

   If yes no. of years

   Whether on treatment - Yes/No

2. HYPERTENSION - (1) Yes (2) No (3Unknown

   If yes no. of years -

Whether on treatment - Yes/No

3. SMOKING - (1) Yes (2) No

If yes no. of years -

How many cigarettes/day -

4. DYSLIPIDEMIA - (1) Yes (2) No
(3)Unknown

5.FAMILY H/O C.A.D – (1) Yes (2)No
(3)Unknown

6.ACID PEPTIC DISEASE 7. STROKE

8.OTHERS

PREVIOUS TREATMENT

EXAMINATION

GENERAL

Consciousness	anemia	cyanosis
clubbing		
Pedal edema	dyspnea	cold extremities
arcus senilis	Xanthelasma	others

VITAL SIGNS

Pulse Blood pressure respiratory rate

Temperature

CARDIOVASCULAR EXAMINATION

Jugular Venous Pulsation Elevated or not

Apical impulse

AUSCULTATION S1 S2

Apex

Tricuspid area

Pul. Area

Aortic area

S3 S4 1.Yes 2.No

Murmur

<u>OTHER SYSTEM</u>

<u>RESPIRATORY SYSTEM</u>

Breath Sounds Creps Rhonchi

ABDOMEN

Hepatomegaly Bruit

<u>COMPLICATIONS</u>

Circulatory failure Killip's class I, II, III,IV

Arrhythmia

INVESTIGATIONS

HB BT CT Bl Gp

FBS PPBS Total Cholesterol

TAG LDL HDL

CHEST X RAY:

ELECTROCARDIOGRAM

site of MI Q ST T Changes

Arrhythmia Chamber hypertrophy/Enlargement

TREATMENT

REINFARCTION

FOLLOW UP ECHOCARDIOGRAPHY

EF LVD LVS Diastolic Dysfn

Regional Wall Motion Abnormality

Hypertrophy/Enlargement

Complications MR VSR Effusion

Clot

DEATH

MASTER CHART

MASTER CHART

GROUP I (MI IN < 40 Yrs)

S.No	Age	Sex	OCC	BMI	DM	SHT	Cig	FH	SITE	TW	PR	SBP	DBP	ST↑	Killip Class	FBS	PPBS	TC	TAG	LDL	HDL	EF	Wall Motion	Reinf arction	DEATH
1	26	M	3	27	-	-	9	+	AW	5	88	180	94	12	I	72	126	120	88	84	45	70	+	-	-
2	32	M	5	23.4	-	+	18	-	AW	4	92	146	92	6	I	68	118	134	92	120	44	35	+	-	-
3	36	F	4	28.5	-	+	0	-	IW	6	87	158	110	4	I	97	112	254	187	132	38	67	+	-	-
4	32	M	2	29.3	-	-	25	+	AW	3.5	94	120	82	5	I	83	126	167	110	78	48	72	+	-	-
5	28	M	3	33.7	-	-	12	+	AW	2	110	110	72	6	I	64	109	145	91	98	45	75	-	-	-
6	39	M	3	30.4	-	-	18	+	ILW	0.5	64	128	84	7	I	98	132	123	76	104	46	65	-	-	-
7	31	M	1	29.0	-	-	10	-	AW	1	92	130	88	8	I	76	124	154	95	178	49	66	+	-	-
8	35	M	3	35.3	-	-	21	+	IW	7	71	96	54	11	I	99	138	198	102	96	51	72	+	-	-
9	40	M	3	32.1	-	-	39	-	IW&R	4	56	80	42	12	I	76	142	267	180	88	30	68	-	-	-
10	33	M	3	29.9	+	-	28	-	EAW	5	83	154	94	10	I	212	320	161	176	175	36	59	+	-	-
11	24	M	5	37.6	-	_	6	+	ILW	6	84	124	74	9	2	83	126	246	234	156	42	67	-	-	-
12	37	M	3	27.9	-	+	22	+	HL	1	78	160	96	8	I	91	143	186	123	150	44	40	+	-	+
13	39	M	3	38.4	-	-	42	-	AW	6	112	130	82	7	I	160	210	193	227	98	48	64	-	-	-
14	29	M	5	31.2	-	-	14	-	AW	2	120	128	78	6	I	88	156	146	78	104	49	66	+	-	-
15	36	M	3	34.2	-	-	9	-	AW	3	98	110	68	8	I	79	127	187	65	99	50	59	+	-	-
16	33	M	3	36.8	-	-	18	+	EAW	5	100	132	74	9	I	70	134	239	167	67	40	70	-	-	-
17	28	M	3	27.5	+	-	0	+	IW	6	71	100	62	4	I	148	240	129	198	78	48	38	-	+	-
18	37	M	3	39.4	-	-	29	-	AW	4	88	150	80	10	I	91	145	121	145	95	39	78	+	-	-
19	39	M	2	38.4	-	+	0	+	APL	1	74	148	94	14	2	83	115	236	102	108	41	65	-	-	-
20	23	M	3	30.6	-	-	12	+	AW	2.5	82	148	76	15	I	86	110	130	114	56	44	60	-	-	-
S.No	Age	Sex	OCC	BMI	DM	SHT	Cig	FH	SITE	TW	PR	SBP	DBP	ST↑	Killip Class	FBS	PPBS	TC	TAG	LDL	HDL	EF	Wall Motion	Reinf arction	DEATH
21	3o	M	3	37.4	-	-	32	+	AW	1.5	88	130	76	5	I	65	112	134	98	84	41	60	+	-	-
22	35	M	3	31.3	-	-	16	+	IPL	4	92	110	66	6	I	78	136	136	76	145	48	67	-	-	-
23	34	M	2	29.7	-	-	15	+	AW	2	93	120	76	9	I	96	142	169	65	120	52	71	-	-	-
24	38	M	5	38.9	-	-	28	-	AW	6	84	114	76	4	I	86	116	175	124	68	42	72	-	-	-
25	40	F	2	36.4	-	+	0	+	IW	3	78	160	112	10	I	67	108	143	165	78	58	65	+	-	-
26	31	M	3	35.0	-	-	24	+	AW	4	88	130	86	11	I	95	122	180	102	138	44	59	+	-	-
27	37	M	3	30.1	+	-	32	+	AW	2	92	150	74	9	I	132	198	220	109	140	50	58	+	-	-
28	36	M	3	29.3	-	-	12	-	IW&RV	3	46	88	56	7	3	77	120	132	143	180	42	72	+	-	-
29	38	M	1	28.5	-	+	18	+	AW	4.5	112	170	98	5	I	98	142	140	128	55	40	64	-	-	-
30	27	M	2	37.5	+	-	8	+	EAW	6	92	108	58	9	4	156	302	287	286	76	30	49	-	-	-
31	30	M	2	34.9	+	-	0	+	HL	1	98	132	78	4	I	137	276	243	298	114	26	45	+	-	-
32	39	M	3	35.6	-	-	24	-	IL	1	76	128	74	8	I	79	154	125	82	130	41	69	+	-	-
33	37	M	3	33.7	-	-	32	+	AW	3	68	124	80	10	I	71	116	172	71	96	45	72	+	-	-
34	38	M	3	40.1	-	+	38	+	AW	5	92	130	90	11	I	69	107	134	96	88	48	59	+	-	-
35	35	M	1	38.2	-	-	12	-	AW	6	80	120	76	12	I	84	124	164	159	140	46	67	-	-	-
36	40	M	5	39.2	-	-	22	-	AW	3	68	110	60	6	I	83	139	257	212	77	51	70	+	-	-

GROUP II (MI IN >40YRS)

S.No	Age	Sex	OCC	BMI	DM	SHT	Cig	FH	SITE	TW	PR	SBP	DBP	ST↑	Killip Class	FBS	PPBS	TC	TAG	LDL	HDL	EF	Wall Motion	Reinf arction	DEATH
41	50	M	2	38.3	+	-	42	+	I&RW	2.5	94	88	46	8	3	320	412	348	161	I23	41	50	-	-	-
42	60	M	5	29.5	-	-	28	-	AW	4	104	120	76	10	2	73	126	132	195	99	43	52	+	-	-
43	60	F	4	42.3	-	+	0	-	AW	3	114	140	88	5	I	79	142	154	102	112	33	42	+	-	-
44	45	M	3	38.6	+	+	0	+	IW	5	89	110	84	6	I	214	312	235	176	120	39	37	+	-	-
45	45	M	3	36.5	-	-	12	-	IW	2.5	98	100	80	9	2	88	165	124	123	116	52	49	-	-	-
46	50	F	4	35.2	+	+	0	-	IPL	6	102	140	80	4	I	164	21	276	236	140	30	40	+	-	-
47	75	M	3	38.3	-	+	0	-	AW	4	105	110	82	8	I	97	154	124	90	140	32	52	+	-	-
48	50	M	1	37.7	-	-	24	+	AW	5	114	130	86	6	I	91	102	132	79	100	46	55	+	+	+
49	65	M	5	34..5	+	-	0	-	EAW	0.5	88	150	102	9	I	187	210	187	156	108	44	42	+	-	-
50	48	M	3	33.5	+	-	38	-	IW&RV	6	70	88	50	4	I	198	298	235	167	148	34	58	-	-	-
51	70	F	4	29.3	+	+	0	-	AW	2	96	160	98	5	I	143	234	187	121	101	32	42	-	-	-
52	52	F	1	38.9	+	+	0	+	AW	7	64	170	102	6	I	186	254	124	95	98	39	49	-	-	-
53	54	M	3	38.5	+	-	0	-	AW	4	80	118	98	4	I	134	315	190	82	132	50	54	+	-	-
54	65	M	3	33.6	-	-	28	-	IW	3	80	110	70	9	I	82	128	241	183	95	42	55	+	-	-
55	50	M	3	36.8	-	-	32	-	AW	3	118	128	72	6	2	68	120	150	109	135	56	61	-	-	-
56	55	F	2	40.5	-	+	0	-	IPL	4.5	90	122	78	8	I	143	164	124	73	96	51	43	+	-	-
57	65	M	5	35.6	+	-	12	+	AW	2	76	140	88	10	I	125	226	138	77	140	46	33	+	+	-
58	57	F	4	29.7	-	-	0	-	EAW	1	94	130	80	4	2	87	135	127	80	119	29	55	+	-	-
59	59	M	3	28.9	-	-	10	-	IL	1	100	112	88	5	I	94	110	199	104	113	44	60	-	-	-
60	52	M	3	34.5	+	+	8	-	IW	2	110	110	72	8	I	176	345	250	189	168	38	43	+	-	-
S.No	**Age**	**Sex**	**OCC**	**BMI**	**DM**	**SHT**	**Cig**	**FH**	**SITE**	**TW**	**PR**	**SBP**	**DBP**	**ST↑**	**Killip Class**	**FBS**	**PPBS**	**TC**	**TAG**	**LDL**	**HDL**	**EF**	**Wall Motion**	**Reinf arction**	**DEATH**
61	60	F	4	37.3	+	-	0	+	AW	2	106	120	94	6	I	132	198	228	324	128	30	58	-	-	-
62	65	M	2	35.4	-	+	37	-	AW	3.5	84	140	92	12	I	92	140	165	142	88	45	54	-	-	-
63	74	M	5	37.5	-	-	0	+	IW	4	60	120	68	8	2	84	126	176	121	92	43	55	+	-	-
64	60	M	2	38.4	+	+	0	-	IPL	4	80	150	98	12	I	154	265	248	188	112	57	39	+	-	-
65	58	F	4	39.5	-	+	0	-	AW	1	96	132	80	4	I	98	132	235	253	94	35	35	+	+	+
66	46	M	3	34.5	+	-	41	-	AW	1	84	148	94	7	2	134	243	256	245	125	38	48	+	-	-
67	70	M	5	35.7	-	-	21	+	AW	3	98	140	94	10	I	76	154	135	102	120	43	54	+	-	-
68	68	F	4	29.9	-	-	0	-	IL	2	82	90	52	9	I	64	132	142	98	86	44	48	-	-	-
69	60	M	1	43.5	+	+	0	-	AW	3	70	130	88	12	I	270	302	132	68	69	46	40	-	-	-
70	48	M	2	37.3	-	-	24	-	IPL	1	54	90	70	5	2	88	168	167	104	84	49	60	+	-	-
71	53	M	3	36.5	+	-	12	+	IW&RV	1	86	160	100	6	I	110	321	208	189	98	45	38	+	-	-
72	49	M	3	39.1	+	-	8	-	AW	5	110	130	92	9	I	137	213	298	298	180	50	45	+	-	-
73	60	F	4	37.8	-	+	0	-	EAW	4	106	120	90	11	4	105	143	139	108	112	37	58	_	-	-
74	42	M	3	32.4	+	+	0	-	IW	3	74	122	98	13	I	125	247	143	187	120	44	54		-	-
75	47	M	3	37.4	-	+	32	-	IW	3.5	90	80	48	4	I	99	176	320	239	124	49	32	+	+	+
76	60	M	2	35.7	+	-	12	-	AW	4	100	150	80	6	I	184	356	243	224	140	41	38	+	-	-

KEY TO MASTER CHART

- TW - Time Window
- DM - Diabetes Mellitus
- SHT - Systemic Hypertension
- Cig - Cigarette smoking in pack years
- FH - Family History of IHD
- Occu - Occupation
- PR - Pulse rate

- SBP - Systolic BP
- DBP - Diastolic BP
- ST ↑ - ST segment elevation
- EF - Ejection Fraction

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